UNIVERSITY OF CALIFORNIA

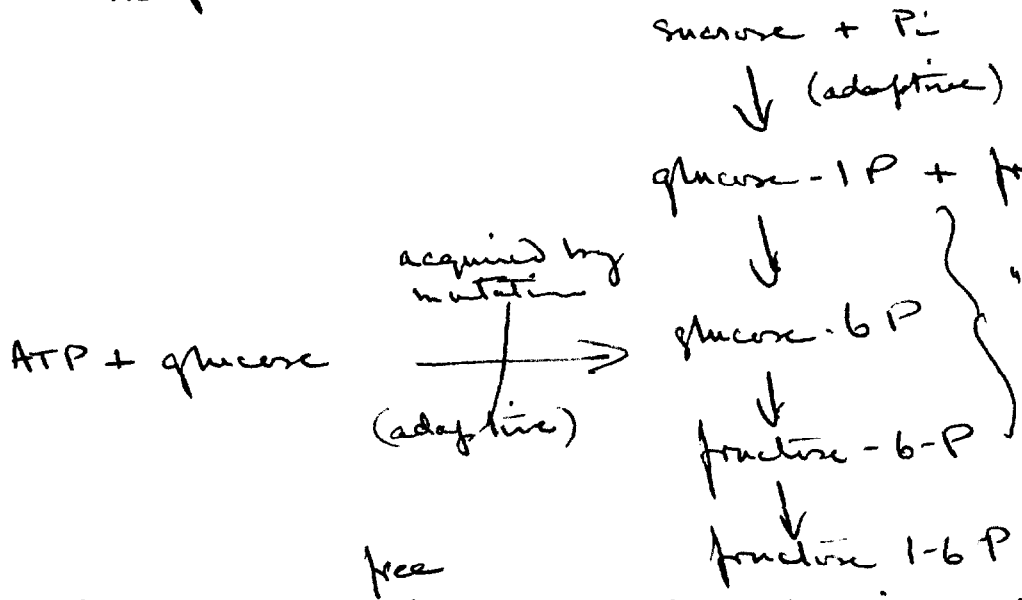
DEPARTMENT OF BACTERIOLOGY
BERKELEY 4, CALIFORNIA

June 2 1950

Dear Joshua —

I enquired at the housing bureau today. Nothing has been found for you yet, but the woman there is confident that there will be no difficulty, so don't worry.

Am very busy getting things cleared up so that I can leave by June 14 for Seattle. However, I wanted to tell you of some fascinating observations by Klein on adaptation in P.s. putrefaciens. Wild type P.p. grows well on sucrose & maltose, but not on glucose or fructose. Last year, Klein showed that it gives rise to spontaneous glucose-using mutants, the biochemical basis of the mutation apparently being the ability of the mutants to produce an (adaptive) glucokinase. The picture then became:



The fate of the fructose moiety is still unclear (no fructokinase in the cells).

Recently, he started to study maltose breakdown (also adaptive) and discovered to our joint amazement that maltose-grown wild-type cells are simultaneously adapted to both maltose and glucose. It could be argued, of course, that growth on maltose had selected for the glucose mutant, but this was eliminated (a) by showing that maltose-grown cells, transferred to glucose, showed the typical 4–5 day lag before growth occurred that signalizes selection

of the rare glucose mutants and (b) that wild-type cells grown in both + then adapted to maltose in resting cell suspension could oxidize glucose + maltose at the same rate thereafter. This finding, of course, wrecks our simple picture of the biochemical basis for the glucose mutation. We have two hypotheses: (a) the Kleinian — that w.t. cells are impermeable to free glucose, so that only glucose formed internally (e.g. in maltose breakdown) can initiate adaptation. On this basis, the mutation would be one involving only a change in permeability. (b) The Stanierian — That for some reason in w.t. cells induction of glucokinase formation by the substrate is blocked, but induction by maltose can occur. This would make the glucose mutation a change in the inductive mechanism, rather than a change in permeability.

Any ideas on this? The Kleinian hypothesis is untenable, I think.

Best wishes

Roger